Doppler Studies of Vena Cava Flows in Human Fetuses

Insights Into Normal and Abnormal Cardiac Physiology

Kathryn L. Reed, MD, Christopher P. Appleton, MD, Caroline F. Anderson, RDMS,
Lewis Shenker, MD, and David J. Sahn, MD

We examined vena cava Doppler flow velocity tracings from 69 fetuses between 22 and 40 weeks’ gestation. Twenty-three fetuses had arrhythmias. Fifteen fetuses had absent end-diastolic Doppler velocities in the umbilical artery, a condition associated with intrauterine growth retardation, and 15 normal fetuses with normal umbilical artery Doppler velocity ratios were matched by gestational age. In studies in 16 additional fetuses, inferior vena cava Doppler velocity waveforms were compared with superior vena cava Doppler velocity waveforms. Peak velocities and time-velocity integrals of forward or reverse flow during systole, early diastole, and atrial contraction were measured. In addition, the time-velocity integral during flow coincident with atrial contraction (a wave) was expressed as a percent of the time-velocity integral of total forward flow during both systole and early diastole. Systolic-to-diastolic ratios of inferior vena cava forward time-velocity integrals were not significantly different from systolic-to-diastolic ratios of superior vena cava forward time-velocity integrals (p=0.86), but the percent of blood moving in a reverse direction during atrial contraction in the inferior vena cava was greater than the percent of blood moving in a reverse direction in the superior vena cava (p<0.05). Relative forward flow in early diastole in the group of normal fetuses increased with advancing gestational age (r=0.60, p<0.05). During premature atrial contractions flow in the inferior vena cava was reversed, and the percent of reverse flow during atrial systole increased significantly from 4.5±0.3% to 28.3±3.7% (mean±SEM, p<0.001). During tachycardias the percent of reverse flow during atrial contraction also increased, with greater increases associated with higher heart rates. During bradycardias the percent of reverse flow during atrial contraction varied with the type of arrhythmia; reverse flow increased with decreasing heart rate in the fetuses with sinus bradycardia. In the group with absent end-diastolic Doppler velocities in the umbilical artery and intrauterine growth retardation, systolic-to-diastolic ratios of the inferior vena cava forward time-velocity integrals increased from 2.76±0.17 to 6.96±0.84 (p<0.001), and the percent of blood moving in a reverse direction during atrial contraction increased from 4.7±0.9% to 13.8±2.3% (p<0.01). These results suggest that fetal vena cava flow velocity patterns are altered in the presence of conditions associated with fetal morbidity and that these alterations can be easily identified by means of Doppler ultrasound techniques. (Circulation 1990;81:498–505)

Cava velocities, along with intracardiac transvalvar velocities, may be obtained non-invasively with Doppler ultrasound.\(^1,2\) Since fetal oxygenation depends on blood returning from the placenta via the inferior vena cava to the fetal heart, alterations in the pattern of vena cava flow have the potential to affect fetal condition. Because previous studies in both animals and humans have demonstrated that alterations in central venous blood velocity patterns accurately reflect abnormalities in cardiac hemodynamics,\(^1,3\) we hypothesized that, in the presence of altered fetal cardiovascular

---

All editorial decisions for this article, including selection of reviewers and the final decision, were made by a guest editor. This procedure applies to all manuscripts with authors from the University of California San Diego or UCSD Medical Center.

From the Department of Obstetrics and Gynecology and the Department of Internal Medicine (C.P.A.), Arizona Health Sciences Center, Tucson, Arizona, and the Department of Pediatrics

(Cardiology) (D.J.S.), University of California San Diego, San Diego, California.

Supported in part by a grant from the American Heart Association, Arizona Affiliate.

Address for correspondence: Kathryn L. Reed, MD, Department of Obstetrics and Gynecology, Arizona Health Sciences Center, 1501 North Campbell Avenue, Tucson, AZ 85724.
hemodynamics such as occur during arrhythmias and with fetal growth failure, cava flows would change, with variations of velocity distribution during systole and early diastole and with atrial contraction. Therefore, we investigated normal patterns and the potential use of Doppler ultrasound studies of human fetal vena cava velocities in the identification of alterations in fetal circulation.

Methods

Fetuses with cardiac arrhythmias, suspected anomalies, or suspected intrauterine growth failure underwent fetal cardiovascular evaluation with two-dimensional and Doppler ultrasound. If an arrhythmia was present, M-mode ultrasound was also performed. The experimental protocol for the Doppler studies was approved by the institutional review board at the Arizona Health Sciences Center, and informed consent was obtained from participating mothers.

Two-dimensional ultrasound examinations were performed to confirm normal cardiac anatomy. Gestational age was estimated on the basis of menstrual weeks and previous ultrasound examinations. Pulsed wave Doppler ultrasound studies of the umbilical artery were performed in each fetus with a 3.5-MHz transducer on a single gate spectral Doppler system with spatial peak temporal average power output less than 100 mW/cm². All Doppler recordings used for measurement were obtained in the absence of fetal breathing.

Figure 1. Two-dimensional Doppler flow velocity waveform of fetal inferior vena cava in normal fetus. Panel A: Two-dimensional ultrasound image of the inferior vena cava; the Doppler gate is immediately proximal to the right atrium. Panel B: Diagram of the image in Panel A. Panel C: Doppler flow velocity waveform from the fetal inferior vena cava. S, systole; D, diastole; a, atrial contraction.

Figure 2. Panel A: Doppler flow velocity waveforms in superior vena cava (SVC) and ascending aorta (AO). Note the timing of onset of the SVC waveform compared to onset of the ascending aorta waveform. Panel B: Doppler flow velocity waveforms in the fetal inferior vena cava (IVC) and the descending aorta (AO). Note the timing of onset of the IVC waveform compared with onset of the descending aorta waveform. The arrow indicates a premature atrial contraction, with no diastolic peak on the IVC tracing, and early systole during the following beat in both the aorta and the inferior vena cava. The diastolic time-velocity integral of the following IVC waveform is increased compared with the other beats. S, systole; D, diastole.
Pulsed wave Doppler studies of the fetal inferior vena cava and, in 19 cases, the superior vena cava were performed (Figures 1 and 2). For the inferior vena cava studies the sample volume (2 mm) was placed under two-dimensional guidance immediately proximal to the right atrium while a sagittal view was imaged that included the fetal right atrium, right ventricle, and ascending aorta. In 27 studies the sample volume was enlarged to 9–20 mm to include the descending aorta so that simultaneous tracings could be obtained from both vessels to confirm the timing of systolic and diastolic events since electrocardiograms were not available (Figure 2). For the superior vena cava examinations either a sagittal or a five-chamber view of the heart was utilized with the sample volume placed to the right of the aorta. Since angles between the ultrasound beam and the estimated direction of blood flow were often more than 30°, actual velocities were not calculated. However, we attempted to quantify the relation of systole and diastole and forward compared to reverse flow events, for all of which the angulation error would be consistent.

Strip-chart recordings of Doppler velocities were obtained at 25 and 50 mm/sec. A digitizing board was used to measure time intervals for heart rate, peak velocity during systole and diastole, and time-velocity integrals. Heart rates were calculated from peak systole to peak systole. Modal velocities were used to measure peaks or time-velocity integrals. A ratio of systolic peak velocity (S) to end-diastolic velocity (D) in the umbilical artery was calculated (S/D ratio). In the inferior vena cava measurements were made of time-velocity integrals and peak velocities during systole and early diastole and during the reverse velocity wave coincident with atrial contraction. A ratio between peak forward velocity during systole (S) and peak forward velocity during early diastole (D) was calculated (peak S/D ratio). For purposes of analysis the waveform occurring during ventricular systole was measured as a single unit although the velocity actually begins during atrial relaxation. Time-velocity integrals (TVI) of flow during systole and early diastole were also compared, using a ratio (TVI S/D ratio). Time-velocity integrals during reverse flow coincident with atrial contraction (a wave) were expressed as a percent of total forward flow during systole added to the following early diastole.

Comparison of Inferior Vena Cava With Superior Vena Cava Waveforms

To demonstrate the correlation between inferior vena cava and superior vena cava flow velocity waveforms, ratios of time-velocity integrals of flow during systole and early diastole from each location were compared. The percent of blood moving in a reverse direction during atrial contraction also was compared in the two vessels.

Arrhythmias

Studies performed during premature atrial contractions were assessed in a similar fashion with the fetus acting as its own control. Waveforms obtained during the normal beat, the premature beat, and the postextrasystolic beat were compared.

Studies were performed during episodes of supraventricular tachycardia. In these cases values obtained during supraventricular tachycardia were compared with values obtained during normal sinus rhythm in the same fetuses.

Studies performed during sinus bradycardia (<120 beats/min) were compared with those obtained from the same fetus during normal sinus rhythm (heart rate, 120–160 beats/min). Values from fetuses with complete heart block were compared with those obtained from fetuses with normal sinus rhythm.

Absent End-Diastolic Umbilical Artery Doppler Velocity/Growth Failure

Fetuses with absent end-diastolic velocities in the umbilical artery were matched by gestational age with fetuses with normal ratios of systolic-to-end-diastolic velocities in the umbilical artery. In this study all fetuses with absent end-diastolic velocities in the umbilical artery had intrauterine growth retardation confirmed at birth, defined as birth weight less than the 10th percentile for gestational age. Inferior vena cava peak velocities, time-velocity integrals, and percent of reverse flow during atrial contraction were compared in the two groups.

Statistical Methods

Paired t tests were used to compare measurements from the inferior vena cava with those from the superior vena cava. Paired t tests were also used to compare measurements made during arrhythmias with measurements made during normal sinus rhythm in the same fetus. Unpaired t tests were used to compare results obtained from fetuses with complete heart block with results obtained from fetuses with normal sinus rhythm since the fetuses with congenital heart block had no episodes of normal heart rates for comparison. Unpaired t tests were used to compare measurements made in the group of fetuses with normal umbilical artery Doppler velocity ratios with measurements made in fetuses with absent end-diastolic velocities in the umbilical artery. Linear regression analysis was used to correlate inferior vena cava measurements with superior vena cava measurements. Linear regression analysis was also used to compare the TVI S/D ratio with gestational age in the fetuses with normal umbilical artery Doppler velocity ratios. In addition, linear regression analysis was used to compare the percent of reverse flow during atrial contraction with the fetal heart rate.

Results

Normal cava blood velocities in the human fetus occur in three phases (Figure 1). The first begins in
late diastole during atrial relaxation and continues through ventricular systole. The second occurs during early diastole. A third brief peak appeared in 87% of the fetuses examined, consisting of flow in the reverse direction during atrial contraction.

Ratios of time-velocity integrals of flow during systole to time-velocity integrals of flow during early diastole decreased during advancing gestation in the 15 normal fetuses ($r = -0.60, p < 0.05$). The percent of reverse flow during atrial contraction in these fetuses was 4.7±0.9%.

Timing of flow during systole, diastole, and atrial contraction was apparent from simultaneous waveforms obtained from both the aorta and the superior or inferior vena cava (Figure 2). Fetal heart rate in the normal group was 146±11 beats/min.

**Inferior and Superior Vena Cava Measurements**

In 16 fetuses ratios of time-velocity integrals during systole and early diastole were compared in the inferior and superior vena cava. In the inferior vena cava the average ratio was 3.32±0.20, which is not significantly different from the ratio in the superior vena cava of 3.70±0.39 ($p = 0.86$). The two ratios correlated significantly ($r = 0.60, p < 0.02$). Reverse flow during atrial contraction, expressed as a percent of total forward flow during systole and early diastole, was significantly greater in the inferior vena cava (8.6±1.3%) than in the superior vena cava (4.9±0.7%, $p < 0.05$).

**TABLE 1. Changes in Inferior Vena Cava Doppler Flow Velocity Waveforms During Premature Atrial Contractions**

<table>
<thead>
<tr>
<th></th>
<th>TVI S/D</th>
<th>Atrial contraction (% forward flow)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal beat</td>
<td>3.31±0.21</td>
<td>4.5±0.3</td>
</tr>
<tr>
<td>Premature beat</td>
<td>---</td>
<td>28.3±3.7*</td>
</tr>
<tr>
<td>PESB</td>
<td>1.96±0.25*</td>
<td>8.2±0.8*</td>
</tr>
</tbody>
</table>

Values are given as mean±SEM. TVI, time-velocity integral; S/D, systolic-to-diastolic ratio; PESB, postextrasystolic beat. *$p<0.001$ compared with normal beat.

**Arrhythmias**

Twenty-three fetuses between 26 and 40 weeks' gestation were examined. Thirteen fetuses had premature atrial contractions. Four fetuses had tachyarrhythmias, two with atrial flutter and two with supraventricular tachycardia. Six fetuses had bradyarrhythmias, three with complete heart block and three with sinus bradycardia.

**Premature Atrial Contractions.** During premature atrial contractions vena cava flow reversed, and the percent of flow during atrial contraction increased significantly (Figure 3 and Table 1) compared with normal sinus beats. During the postextrasystolic beat the proportion of blood flow during early diastole increased compared with normal with a resultant decrease in the TVI S/D ratio. The percent of reverse flow during atrial contraction decreased compared with the premature beat but remained increased compared with the normal beat (Table 1).

**Tachyarrhythmias.** During tachyarrhythmias ($n=4$) the percent of reverse flow during atrial contraction increased (38.0±10.8%) compared with normal heart rates (6.5±1.4%, $p < 0.05$) in the same fetuses (Figure 4). The percent of reverse flow increased with increasing heart rate ($r = 0.96, p < 0.01$, slope = 0.4). At faster heart rates time-velocity integrals during systole and early diastole merged so that ratios could not be calculated. Ventricular rates in this group ranged from 180 (atrial flutter) to 238 beats/min (average, 205.0±12.1 beats/min).

**Bradycardia.** Fetuses with sinus bradycardia ($n=3$) maintained a normal ratio of systolic-to-diastolic flow (2.7±0.8, $p=NS$) and increased the percent of reverse flow during atrial contraction (14.0±2.1%,

---

**FIGURE 3.** Doppler flow velocity waveform in fetal inferior vena cava during premature atrial contraction. Diastolic flow is interrupted by reverse flow during the atrial contraction (arrow). During the following beat diastole (D) is prolonged, the diastolic time-velocity integral is increased compared with the other two beats, and reverse flow during atrial contraction is increased compared with normal (see text and Table 1). S, systole; D, diastole; a, atrial contraction.

**FIGURE 4.** Doppler flow velocity waveforms in inferior vena cava during normal sinus rhythm (Panel A) and supraventricular tachycardia (Panel B) in the same fetus. Note the increase in reverse flow with atrial contraction (a) during the rapid heart rate (180 beats/min).
p<0.02, Figure 5 and Table 2). The percent of reverse flow during atrial contraction increased with decreasing heart rate \((r=-0.99, p<0.001, \text{slope}=-0.7)\). Heart rates in this group ranged from 100 to 119 beats/min (average, 109.3±9.5).

Fetuses with complete heart block \((n=3)\) had markedly increased proportions of forward flow during diastole \((TVI/S/D \text{ ratio, } 0.50±0.05, p<0.001, \text{compared with the normal beats in the group with premature atrial contractions})\) and had a normal percentage of reverse flow during atrial contraction \((5.4±1.0\%, p=NS, \text{Figure 6 and Table 2})\). Atrial rates ranged from 80 to 150 beats/min; ventricular rates ranged from 50 to 54 beats/min. Reverse flow during ventricular contraction was not measured; reverse flow with atrial contraction did not always occur during the prolonged diastole.

**Absent End-Diastolic Umbilical Artery Doppler Velocities/Growth Failure**

Thirty fetuses between 25 and 36 weeks of gestation were examined. Fifteen fetuses had absent end-diastolic velocities in the umbilical artery (“no flow”), and 15 fetuses matched by gestational age had normal umbilical artery waveforms (Table 3). All fetuses with absent end-diastolic velocities in the umbilical artery had intrauterine growth retardation, with birth weight less than the 10th percentile for gestational age.

**TABLE 2. Changes in Inferior Vena Cava Doppler Flow Velocity Waveforms During Fetal Bradycardias**

<table>
<thead>
<tr>
<th></th>
<th>TVI S/D</th>
<th>Atrial contraction (% forward flow)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus bradycardia</td>
<td>2.7±0.8</td>
<td>14.0±2.1†</td>
</tr>
<tr>
<td>Normal beat/ SB</td>
<td>3.2±0.7</td>
<td>5.2±0.9</td>
</tr>
<tr>
<td>Normal beat/ PAC</td>
<td>3.3±0.2</td>
<td>4.5±0.3*</td>
</tr>
<tr>
<td>Complete heart block</td>
<td>0.5±0.1‡</td>
<td>5.4±1.0</td>
</tr>
</tbody>
</table>

Values are given as mean±SEM. TVI, time-velocity integral; S/D, systolic-to-diastolic ratio; normal beat/ SB, normal heart rates in fetuses with sinus bradycardia (SB); normal beat/PAC, normal heart rates in fetuses with premature atrial contractions (PAC).

†p<0.01 compared with atrial contraction in sinus bradycardia group.

†p<0.02 compared with normal rate in same fetus.

‡p<0.001 compared with normal rate, PAC group.

In the abnormal group the ratio of peak velocity during systole to peak velocity during early diastole and the ratio of time-velocity integrals during systole to time-velocity integrals during early diastole were increased significantly compared with normal. The percent of reverse flow during atrial contraction was increased significantly compared with normal (Figure 7 and Table 3). Heart rates were similar in both groups (normal, 146±11 beats/min; no flow, 144±12 beats/min).

**Discussion**

Pulsed Doppler ultrasound has been used to examine the cardiovascular system of the human fetus to establish normal values and to study the physiologic changes that accompany the development of abnormalities such as cardiac arrhythmias and growth failure. Prior studies of the human fetus have shown that right ventricular volume flow is 30% greater than left ventricular volume flow, and that the Frank-Starling mechanism is operative, and that mean intracardiac velocities decrease in the presence of supraventricular tachycardia. In fetuses with absent end-diastolic Doppler velocities in the umbilical artery, right ventricular volume flow has been reported to be increased compared with normal. This report on human fetal inferior and superior vena cava blood velocity waveforms is an extension of intracardiac Doppler velocity studies. In this study we found that cava velocities during diastole and during atrial contraction were abnormal in conditions associated with fetal morbidity.

**TABLE 3. Inferior Vena Cava Doppler Flow Velocities in Fetuses With Normal and Absent End-Diastolic Velocities in Umbilical Artery**

<table>
<thead>
<tr>
<th></th>
<th>IVC S/D (peak)</th>
<th>IVC S/D (TVI)</th>
<th>Atrial contraction (% forward flow)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1.71±0.06</td>
<td>2.76±0.17</td>
<td>4.7±0.9</td>
</tr>
<tr>
<td>“No flow”</td>
<td>2.03±0.12</td>
<td>6.96±0.84</td>
<td>13.8±2.3</td>
</tr>
</tbody>
</table>

Values are given as mean±SEM. IVC, inferior vena cava; S/D, systolic-to-diastolic ratio; TVI, time-velocity integral; “no flow,” absent end-diastolic velocities in the umbilical artery.
Cava velocity waveforms in the adult have been used to identify hemodynamic alterations from normal, including idiopathic congestive cardiomyopathy, restrictive myocardial processes, and coronary artery disease. Normally, forward flow in the superior vena cava in adults occurs in three phases: a single and usually the largest peak begins during atrial relaxation and continues through ventricular systole; a second, usually smaller peak occurs during early diastole; and reverse flow usually occurs coincident with atrial contraction. Animal and human studies have shown the interaction between cardiovascular hemodynamics and changes in cava velocity. Use of norepinephrine in instrumented fetal lambs changed the blood velocity in the superior vena cava, with an increase in amplitude of pulsation of the waveform. Similar effects were seen in the inferior vena cava in the presence of acute hypoxia.

Doppler studies of the superior vena cava during inspiration and expiration have shown variation in the velocity of blood flow. It has been proposed that the superior vena cava waveform during respiration may be a sensitive mechanism by which to assess right ventricular function and pressures in normal and abnormal patients. In the fetus with the patent foramen ovale cava velocities will reflect intracardiac hemodynamics and physiology of both the right and left ventricles.

Studies in the human fetus are limited by the methods available for investigation. Pressure measurements in the fetal cardiovascular system require invasive techniques that are not currently employed. Volume flow measurements may be performed with Doppler ultrasound although errors may be substantial. While Doppler flow velocity measurements may be performed noninvasively, the interpretation of the results must be extrapolated from studies performed in instrumented adults and animals.

In this report inferior vena cava velocity measurements correlated with superior vena cava velocity measurements although the percent of reverse flow with atrial contraction was higher in the inferior vena cava. Despite differences in physiology, an increase in reverse flow with atrial contraction in the inferior vena cava compared with the superior vena cava is also present in adults. Because the inferior vena cava is the primary source of oxygenated blood in the fetus and because reverse flow in the inferior vena cava is greater than in the superior vena cava, most of the studies in this report concentrated on changes in the inferior vena cava Doppler flow velocity waveform.

The ratio of systolic to diastolic flow in the human fetal inferior vena cava (2.8±0.2) is slightly higher than that reported in the human adult superior vena cava (2.2–2.3). The percent of reverse flow during atrial contraction in the human fetal inferior vena cava (4.7±0.9%) is slightly lower than that reported in the human adult superior vena cava (5–6%). These differences may reflect the higher heart rate in the fetus compared with the adult and differing properties of fetal hemodynamics, chamber or myocardial stiffness, or rate of ventricular relaxation compared with the adult.

**Arrhythmias**

In the current work we demonstrated changes in the human fetal vena cava waveform with premature atrial contractions. Increases in the percent of reverse flow with atrial contraction and increases in early diastolic flow during both the premature beat and the postextrasystolic beat were found. With premature atrial contractions forward flow is interrupted by the premature contraction. The premature atrial contraction begins with a flow reversal, which aids in its identification as an arrhythmia of atrial origin. The premature contraction by definition occurs earlier in the cardiac cycle than normal. The premature beat may occur during ventricular diastole, during which the atrium and ventricle normally fill passively from the venae cavae. Increased flow in the reverse direction, from the right atrium into the inferior and superior venae cavae, may occur because the atrium contracts when it is more filled than at end diastole or because the right ventricular pressure is high and the ventricle is not relaxed. If the premature contraction occurs even earlier in the cardiac cycle during ventricular systole when the ativoventricular valves are closed, a “cannon” a wave results so that the velocity of blood in the reverse direction is even greater. During the compensatory postextrasystolic pause following the premature beat, early diastolic velocity increases compared with normal. This increase may occur because with prolonged diastole the ventricle continues to fill and flow into the
ventricle (and atrium) increases proportionately. Alternatively, right ventricular relaxation may be abnormally increased, or right atrial pressure in early diastole may be abnormally high. Atrial contraction still results in increased reverse flow because of overfilling of the atrium during the prolonged diastole.14

During supraventricular tachycardia reverse flow during atrial contraction increased. This increased velocity of reverse flow during atrial contraction may result from overfilling of the atrium due to inadequate ventricular filling time during the rapid heart rate. None of these fetuses had tricuspid regurgitation or hydrops. Reversal of flow into the inferior vena cava has been reported in infants with atrial tachycardia induced during transesophageal pacing.15 The increase in reverse flow may explain the difficulty of maintaining adequate forward flow in the face of persistent supraventricular tachycardia, with the eventual development of hydrops in the fetus.

Although the numbers are small, the two patterns with the different types of bradycardia are interesting. With complete heart block in fetuses, the atrial rate is nearly twice as fast (or greater) than the ventricular rate. The ventricle continues to fill with each atrial contraction, which occurs during ventricular diastole. Reverse flow with atrial contraction during complete heart block was normal. In the fetuses examined, ventricular rates (50 beats/min or more) were in the range normally associated with good fetal outcomes.16 With sinus bradycardia only one atrial contraction occurs for each ventricular beat, and the percent of reverse flow during atrial contraction increases. This difference in reversal of flow may explain why fetuses usually tolerate complete heart block in the absence of cardiac anomalies and why sinus bradycardia at similar ventricular rates is not well tolerated. Sinus bradycardia may result in left ventricular filling with deoxygenated blood. Normally, deoxygenated blood from the superior vena cava streams into the right ventricle and then through the ductus arteriosus into the descending aorta.17 Oxygenated blood from the placenta crosses the right atrium from the inferior vena cava, into the foramen ovale, left atrium, left ventricle, and into the fetal cerebral structures.17 Arrhythmias that interfere with this normal streaming pattern will allow mixture of blood in the right atrium and result in less-oxygenated blood entering the left side of the fetal circulation.

**Absent End-Diastolic Umbilical Artery Doppler Velocities and Growth Failure**

The proportion of blood velocity during end-diastole in the umbilical artery normally increases with advancing gestation,6 possibly because of an increase in placental vascular volume and a decrease in vascular resistance. We have previously shown that fetal morbidity (100%), intrauterine growth retardation (80%), and mortality (42%) are increased when umbilical artery end-diastolic velocities are absent.6

![Figure 8](image-url)

**Figure 8.** Comparison of heart rate with percent of forward flow (% FF) in inferior vena cava which moves in a reverse direction with atrial contraction (AK). Normal fetal heart rate is between 120 and 160 beats/min.

In addition, we have demonstrated that right ventricular volume flow increases in this group of fetuses compared with normal.6 In the current report we show that changes in inferior vena cava velocities occur in fetuses with absent end-diastolic umbilical artery velocities. In the inferior vena cava of these fetuses, all of whom were growth retarded, the proportion of blood flow during diastole decreased, and the percent of blood moving in a reverse direction during atrial contraction increased. The relative decrease in diastolic flow is compatible with a reduced early diastolic pressure difference between the atrium and the ventricle. A decreased rate of ventricular relaxation may explain a low pressure gradient between the right atrium and the right ventricle.18 The increase in the percent of blood moving in a reverse direction during atrial contraction may be due to increased ventricular pressure or to decreased ventricular compliance. These blood velocity changes are strikingly similar to changes seen in instrumented lamb fetuses, in which the amplitude of the cava waveform increases after the administration of norepinephrine or in the presence of hypoxia.3

The most consistent pattern seen in the inferior vena cava velocities of fetuses with clinically important arrhythmias (sustained tachyarrhythmias, sinus bradycardias) and with growth retardation was the increase of reverse flow during atrial contraction. Forward flow of the most highly oxygenated blood returning from the placenta is impeded, resulting in less oxygen availability to the fetal tissues. If this pattern of impaired forward flow is maintained, fetal distress (hypoxia or hydrops) may result. Clinical experience confirms the association of these conditions with poor fetal outcome. It may be that there is an optimal percent of reverse flow with atrial contraction, probably less than 10%, as demonstrated in Figure 8. At heart rates outside the range of normal (120–160 beats/min), the percent of forward flow moving in the reverse direction with atrial contraction increases. Fetuses with absent end-diastolic velocities in the umbilical artery and intruterine
growth retardation also have increased reverse flow with atrial contraction. While the problem in the arrhythmia group may rest with abnormal heart rates, changes in ventricular function in fetuses with growth retardation may accomplish the same result (increased reverse flow) such that fetal hypoxia results from altered forward flow of oxygenated blood.

In conclusion, flow velocity patterns in the fetal vena cava are altered in conditions associated with fetal morbidity. In the presence of specific arrhythmias or growth failure, fetal morbidity appears to be associated with an increase in reverse flow in the inferior vena cava during atrial contraction.

References
8. Schlotterer M, Reed KL, Anderson CF, Shenker L: Doppler studies of umbilical and uteroplacental vasculature in human pregnancy (abstract). American Institute of Ultrasound in Medicine, New Orleans, October 6–9, 1987

KEY WORDS • arrhythmias • fetuses, human • growth
Doppler studies of vena cava flows in human fetuses. Insights into normal and abnormal cardiac physiology.
K L Reed, C P Appleton, C F Anderson, L Shenker and D J Sahn

Circulation. 1990;81:498-505
doi: 10.1161/01.CIR.81.2.498

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/81/2/498